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Lower Dietary Polyunsaturated to Saturated Fat Ratio Is Associated With Increased Visceral Adiposity

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Background: Increased visceral fat is associated with increased insulin resistance and cardiovascular risk factors. While dietary fat is related to cardiovascular risk factors, less is known about the relationship between dietary fat and its components to visceral fat.

Methods: Subjects (n=84, 46 women and 38 men) were aged 55 to 75 years, sedentary, without clinical cardiovascular disease, nonsmoking and without diabetes. Diet was assessed by analyzing 3-day food records. Body composition was assessed with a variety of techniques including anthropometry, total body fat by dual energy X-ray absorptiometry (DEXA), and abdominal fat distribution by MRI.

Results: Among dietary intake, % saturated fat energy ($r=0.23$, $p<0.05$) and polyunsaturated fat / saturated fat ratio (PUFA/SFA ratio) ($r=-0.33$, $p<0.01$) were correlated to abdominal visceral fat. Waist circumference ($r=0.71$, $p<0.001$) and total body fat ($r=0.40$, $p<0.01$) correlated to visceral fat. Men had more visceral fat than women. (162 ± 56 vs. 124 ± 56 cm², $p<0.01$) Age did not correlate to visceral fat. In multivariate analysis, lower PUFA/SFA ratio was independently associated ($p<0.05$) with higher visceral fat after adjustment for gender and total body fat.

Conclusion: A higher dietary consumption of polyunsaturated fat relative to saturated fat was an independent predictor of lower visceral fat. Modification of type of fat intake may have an impact in specifically reducing visceral fat in addition to the well-known beneficial influence to serum lipid profile.

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Post-Prandial Lipid and Lipoprotein Responses in Patients Taking HIV Protease Inhibitors

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Background: The dyslipidemia associated with human immunodeficiency virus protease inhibitors (HIV PIs) is associated with increased levels of triglyceride-rich lipoproteins and cholesterol-rich remnants. The purpose of this study was to determine if use of HIV PIs increases post-prandial lipemia.

Methods: After a 12-hour fast, 39 HIV+ and 10 HIV- euthyroid, non-diabetic subjects ingested an oral lipid load consisting of a milkshake that contained (mean \pm standard error) 101 ± 1 grams of fat. All HIV+ subjects were on stable anti-retroviral regimens; 24 were taking PIs, 15 were not taking PIs. Serum samples were obtained at baseline prior to the fat load, then every 2 hours for 10 post-prandial hours. Lipid mass concentrations of 16 lipoprotein fractions, average particle sizes, and particle concentrations were quantified by nuclear magnetic resonance spectroscopic lipoprotein subclass analysis (LipoScience, Inc., Raleigh, North Carolina).

Results: HIV- subjects were younger (31 ± 3 years, $p<0.001$) than HIV+ subjects taking PIs (43 ± 2 years) or not (41 ± 2 years). ApoE phenotypes were similar in all 3 groups. The age-adjusted areas under the concentration curves (AUCs) for intermediate-density lipoproteins (IDL) were significantly higher for HIV+ subjects taking PIs than for HIV+ subjects not on PIs and HIV- controls ($p<0.005$). The AUC's for triglycerides was higher in subjects on PIs ($p=0.025$), and tended to be higher in HIV+ subjects not on PIs ($p=0.064$), as compared to HIV- controls. AUC's for large very low-density lipoproteins and total cholesterol also tended to be higher in both HIV+ groups than controls ($p<0.08$). In subjects on PIs, increased chylomicrons were observed in the late post-prandial period, suggesting delayed clearance ($p<0.05$).

Conclusion: Individuals with HIV infection have increased post-prandial hypertriglyceridemia. Individuals taking PIs have impaired clearance of IDL. Clearance of chylomicrons may be decreased in individuals taking PIs. Individuals treated for HIV infection, especially those taking PIs, may be at increased vascular risk due to alimentary hyperlipidemia.

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Improved Lipid Profile and Reduced Lipid Peroxidation With Soy Milk in Patients With Primary Hypercholesterolemia

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Background: Soy protein, in addition to ATP III Therapeutic Lifestyle Changes (TLC) diet, has been reported as a therapeutic option to optimize LDL-cholesterol lowering. Therefore, this study was aimed to assess whether the consume of soy milk could add significantly to lipid profile and lipid peroxidation in comparison with low fat cow milk, in patients with primary hypercholesterolemia. **Methods and Results:** A double-blind, randomized, crossover study evaluated 60 men and women with primary hypercholesterolemia under TLC diet for at least six weeks. Lipid profile was obtained at baseline, 6 and 12 weeks with the patients randomly assigned to receive first one liter per day of either soy milk or low fat cow milk. No significant differences in total calories (60 vs. 57 kcal) or cholesterol (0 vs. 1 mg) were identified when comparing the two milk compositions (amount per 200 ml). However, soy milk had higher fat (3.5 vs. 0.01g) and smaller carbohydrate content (1 vs. 8.6 g). Patients were carefully monitored regarding dietary intake.

CHANGES IN LIPIDS AND TBARS

Period of treatment	Total cholesterol (mg/dL)	LDL-cholesterol (mg/dL)	HDL-cholesterol (mg/dL)	TG (mg/dL)	TBARS (nM)
Base line	241 \pm 5	157 \pm 4	58 \pm 2	136 \pm 9	1.8 \pm 0.1
Soy milk	237 \pm 4	148 \pm 4*	62 \pm 2 #	133 \pm 8	1.5 \pm 0.1
Cow milk	240 \pm 4	157 \pm 4	57 \pm 2	134 \pm 9	1.9 \pm 0.1

Values are means \pm SEM; TG = Triglycerides; * $p<0.05$ vs. base line and cow milk; # $p<0.01$ vs. base line and cow milk.

Conclusions: Soy milk improved lipid profile and attenuated lipid peroxidation. Considering the large use of milk in modern societies, this strategy could be suggested in the prevention of cardiovascular disease, particularly in hypercholesterolemic populations.

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Effects of Step I Diet on Nitric Oxide Bioactivity, Oxidant Stress, Inflammation, Plaque Stabilization, and Hemostasis in Hypercholesterolemic Patients With Coronary Artery Disease

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Background: We investigated the effects of American Heart Association Step I Diet on endothelial function- nitric oxide (NO) bioactivity and serological markers of oxidant stress, inflammation, plaque stabilization, and hemostasis.

Methods: We administered American Heart Association Step I Diet during 14 weeks to 50 patients with coronary artery disease. * $P<0.05$; ** $P<0.01$; *** $P<0.001$ vs. Baseline. Data= mean \pm SD.

Results: Diet therapy significantly reduced lipoproteins levels, and improved the percent flow-mediated dilator response (FMD) to hyperemia from 4.46 ± 1.66 to 5.29 ± 1.81 by $25 \pm 36\%$ ($P<0.001$) and increased plasma levels of nitrate from 64 ± 28 to 79 ± 42 μ M by $36 \pm 74\%$ ($P=0.049$), and reduced plasmin levels of malondialdehyde (MDA), a marker of free radical from 1.75 ± 0.71 to 1.52 ± 0.54 μ M by $7 \pm 30\%$ ($P=0.011$). Diet therapy, however, did not significantly change plasma levels of monocyte chemoattractant protein (MCP-1), intercellular adhesion molecule (ICAM-1), and C-reactive protein, and total matrix metalloproteinase (MMP)-9 and MMP-3, and fibrinogen and tissue factor activity and tissue factor pathway inhibitor activity.

Conclusions: Diet therapy improved endothelium-dependent vasodilation with increase of plasma nitrogen oxide and reduction of oxidant stress. However, Diet therapy did not change markers of inflammation, plaque stability, and hemostasis.

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Fluvastatin Prevents Cardiac Events Following Successful Percutaneous Coronary Intervention in Patients With Multivessel Disease: The Lescolfi Intervention Prevention Study

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Background: Patients with multivessel disease have a worse prognosis after percutaneous coronary intervention (PCI) than those with single-vessel disease. This analysis evaluated the impact of early fluvastatin treatment on major adverse cardiac events (MACE) in patients with coronary heart disease and multivessel disease, who had been enrolled into the Lescol[®] Intervention Prevention Study (LIPS), a major prospective randomized clinical trial that investigated the efficacy of fluvastatin after a successful first PCI (1).

Methods: A total of 1,677 patients with stable and unstable angina or silent ischemia were randomized to receive treatment with fluvastatin 80 mg/day (40 mg twice daily) (n=322 with multivessel disease, n=522 with single-vessel disease) or matching placebo (n=292 with multivessel disease, n=541 with single-vessel disease) after their first successful PCI.

Results: In placebo controls with multivessel disease, the incidence of MACE was 33.9% as compared to 22.7% in those with single-vessel disease. Fluvastatin significantly reduced the risk of MACE in patients with multivessel disease by 34% (RR, 0.66; 95% CI, 0.48-0.91; $p=0.01$) compared with placebo; patients with single-vessel disease had a 14% risk reduction (RR, 0.86; 95% CI, 0.66-1.13; $p=0.28$). When restenotic complications in the first 6 months post-PCI were excluded, fluvastatin significantly reduced the risk of MACE by 42% (RR, 0.58; 95% CI, 0.48-0.91; $p=0.002$) in patients with multivessel disease compared with placebo; patients with single-vessel disease had a risk reduction of 23% (RR, 0.77; 95% CI, 0.57-1.03; $p=0.07$).

Conclusion: Fluvastatin significantly reduces the risk of MACE after PCI in patients with multivessel disease compared with placebo.

1. Serruys PWJC, et al. JAMA 2002;287:3215-22.